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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
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10/530,871

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Hans-Georg Kreysch

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7590

03/04/2009

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EXAMINER

HUYNH, PHUONG N

ART UNIT

PAPER NUMBER

1644

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PAPER

Please find below and/or attached an Office communication concerning this application or proceeding.

The time period for reply, if any, is set in the attached communication.

Office Action Summary

Application No.

10/530,871

Applicant(s)

KREYSCH ET AL.

Examiner

PHUONG HUYNH

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1644

Period for Reply -- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE three MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

- 1) ☒ Responsive to communication(s) filed on 05 January 2009.
- 2a) ☐ This action is **FINAL**. 2b) ☒ This action is non-final.
- 3) ☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

- 4) ☒ Claim(s) 30 and 33-40 is/are pending in the application.
- 4a) Of the above claim(s) _____ is/are withdrawn from consideration.
- 5) ☐ Claim(s) _____ is/are allowed.
- 6) ☒ Claim(s) 30 and 33-40 is/are rejected.
- 7) ☐ Claim(s) _____ is/are objected to.
- 8) ☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

- 9) ☐ The specification is objected to by the Examiner.
- 10) ☐ The drawing(s) filed on _____ is/are: a) ☐ accepted or b) ☐ objected to by the Examiner.
Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).
Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).
- 11) ☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

- 12) ☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).
- a) ☐ All b) ☐ Some * c) ☐ None of:
1. ☐ Certified copies of the priority documents have been received.
 2. ☐ Certified copies of the priority documents have been received in Application No. _____.
 3. ☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

- 1) ☐ Notice of References Cited (PTO-892)
- 2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
- 3) ☒ Information Disclosure Statement(s) (PTO-8508)
Paper No(s)/Mail Date 1/5/09
- 4) ☐ Interview Summary (PTO-413)
Paper No(s)/Mail Date _____
- 5) ☐ Notice of Informal Patent Application
- 6) ☐ Other: _____

DETAILED ACTION

1. A request for continued examination under 37 CFR 1.114, including the fee set forth in 37 CFR 1.17(e), was filed in this application after final rejection. Since this application is eligible for continued examination under 37 CFR 1.114, and the fee set forth in 37 CFR 1.17(e) has been timely paid, the finality of the previous Office action has been withdrawn pursuant to 37 CFR 1.114. Applicant's submission filed on January 5, 2009 has been entered.
2. Claims 30 and 33-40 are pending and being acted upon in this Office Action.
3. The Exhibit A listed in PTO 1449 filed January 5, 2009 has been considered but cross-out because it is inappropriate to be printed on the issued patent.
4. The following is a quotation of the second paragraph of 35 U.S.C. 112:
The specification shall conclude with one or more claims particularly pointing out and distinctly claiming the subject matter, which the applicant regards as his invention.
5. Claim 30 is rejected under 35 U.S.C. 112, second paragraph, as being indefinite for failing to particularly point out and distinctly claim the subject matter which applicant regards as the invention.

The phrase “enhanced ErbB1 blocking *and/or inhibition and induction* of down-regulation of ErbB1” in claim 30 is indefinite and ambiguous because inhibition and induction are mutually exclusive. The conjunction “and” between inhibition and induction is improper. Correction is required. It is suggested that Claim 30 be amended to recite “A composition comprising a humanized MAb 425 (h425) or binding fragment thereof and a chimeric MAb 225 (c225) or binding fragment thereof, that bind to different epitopes located within the ErbB1 receptor ligand-binding domain on the same ErbB1 receptor, thereby increasing the amount of antibody bound per receptor and thus per cell by the same antibody dose and causing an enhanced inhibition of EGF binding to ErbB1, enhanced cell aggregation and/or enhanced ErbB1 receptor internalization as compared with a composition comprising only said humanized MAb 425 (h425) or said chimeric MAb 225 (c225).

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6. The following is a quotation of the appropriate paragraphs of 35 U.S.C. 103(a) that form the basis for the rejections under this section made in this Office Action:

A person shall be entitled to a patent unless:

(a) A patent may not be obtained though the invention is not identically disclosed or described as set forth in section 102 of this title, if the differences between the subject matter sought to be patented and the prior art are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains. Patentability shall not be negated by the manner in which the invention was made.

7. This application currently names joint inventors. In considering Patentability of the claims under 35 U.S.C. 103(a), the examiner presumes that the subject matter of the various claims was commonly owned at the time any inventions covered therein were made absent any evidence to the contrary. Applicant is advised of the obligation under 37 CFR 1.56 to point out the inventor and invention dates of each claim that was not commonly owned at the time a later invention was made in order for the examiner to consider the applicability of 35 U.S.C. 103(c) and potential 35 U.S.C. 102(e), (f) or (g) prior art under 35 U.S.C. 103(a).
8. Claims 30, 33-38 and 40 stand rejected under 35 U.S.C. 103(a) as being unpatentable over Ye et al (of record, Oncogene 18: 731-738, 1999; PTO 892) or Mendelsohn et al (newly cited, Oncogene 19: 6550,6565, 2000; PTO 892) each in view of US Pat No 5,558,864 (of record, issued Sept 24, 1996; PTO 892).

Ye et al teach a pharmaceutical composition comprising a first antibody molecule such as human-mouse chimeric anti-EGF receptor mAb C225 that binds to ErbB1 (also known as EGF receptor) and a second antibody molecule such as humanized anti-HER2 mAb 4D5 that binds to (ErbB2) (see entire document, page 732, in particular). Ye et al teach the chimeric antibody C225 competes with EGF for receptor binding and has been shown that repeatedly administered to patient for up to 3 months without eliciting a host anti-antibody response in clinical trial (see page 731, col. 2, second paragraph, in particular). Ye et al teach simultaneous blockade of two related EGF receptors with combination of two antibodies resulted in significant augmentation of growth inhibition of cancer cells (see abstract, page 73, col. 1, FIG 1A, in particular), significant inhibition of cell growth progression by arresting the cell at the G1 stage of cell cycle (see page 732, results, FIG 2, in particular). Ye et al teach it is reasonable to combine monoclonal antibody mAb C225 and mAb 4D5, in order to inhibit the proliferation of cancer cells stimulated by both EGF receptor and HER2 signals since EGF receptors can be activated by either EGF receptor

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homodimerization or heterodimerization with HER2 (see page 736, col. 1, in particular). Ye et al teach binding of two antibodies to both receptors may prevent the formation of active receptor heterodimers such as ErbB1/ErbB2 (see page 737, col. 1, in particular). Ye et al conclude that since both chimeric mAbs C225 and humanized mAb 4D5 are in clinical trials and have demonstrated no severe toxicity, the possibility of combining them in patients should be strongly considered (see page 737, col. 1, in particular). The reference chimeric anti-EGF receptor mAb C225 that binds to ErbB1 (also known as EGF receptor) is identical to the chimeric anti-EGF receptor mAb C225 in the claimed pharmaceutical composition.

Mendelsohn et al teach chimeric MAb 225 (IMC-c225) binds to EGF receptor (also known as ErbB1) with higher affinity ($K_d = 0.39$) than the murine MAb and the natural ligands and the chimeric antibody was produced by obviate the immune response produced in humans by repetitive exposure to treat cancer (see page 6552, col. 1, in particular). Mendelsohn et al teach MAb C225 is acting as a tyrosine kinase inhibitor specific for EGF receptor (see page 6552, col. 2, in particular). Mendelsohn et al teach antibody binding fragment such as $F(ab')_2$ fragment of reference MAb C225 can inhibit xenograft growth (see page 6553, col. 2, (vi), in particular). Mendelsohn et al further teach a pharmaceutical composition comprising a combination of anti-EGFR and anti-ErbB2 mAbs resulted in additive anti-proliferative effects suggesting a potential benefit of this combined therapy in the treatment of human cancers stimulated by EGF receptor (also known as ErbB1) and ErbB2 signal, see page 6554, col. 2, reference cited therein, in particular). Mendelsohn et al further teaches a pharmaceutical composition comprising the reference chimeric MAb 225 (IMC-c225) in combination with conventional cytotoxic agent or chemotherapeutic agent such as cisplatin (CTP-11 (see page 6556, col. 1, second full paragraph, in particular), doxorubicin, paclitaxel (see page 6560, col. 1, in particular) or tyrosine kinase inhibitor such as ZD1839 (see page 6558, col. 2, in particular). Mendelsohn et al teach in general, antibody approach ensures complete specificity for the EGFR kinase. Furthermore, the MAb treatment results in receptor down regulation, which could be advantageous. MABs have less capacity to reach normal intestinal epithelium, which appears to be an advantage for monoclonal mediated EGF receptor blockade therapy since diarrhea was a dose-limiting toxicity with the oral kinase inhibitor but was not observed with the MAB.

The invention differs from the teachings of the references only in that the pharmaceutical composition wherein the humanized monoclonal antibody is MAb 425 (h425) that binds to a

different epitope on the ErbB1 receptor instead of humanized anti-HER2 mAb 4D5 that binds to (ErbB2) as taught by Ye et al or Mendelsohn et al.

However, the '864 patent teaches a pharmaceutical composition comprising a humanized monoclonal antibody such as h425 or binding fragment thereof such as F(ab')₂ that binds to EGF receptor (also known as ErbB1) derived from murine monoclonal antibody MAb 425 for treatment of human tumor (see entire document, col. 1, line 65-66, col. 2, line 57, col. 4, lines 43, col. 21, lines 1-37, claims of the '864 patent, col. 21, lines 60-67, in particular). The '864 patent teaches the advantages of humanized antibody h425 is that the humanized antibody is less likely to raise an anti-antibody immune response in humans and the Mab h425 has a longer half-life in humans (see col. 22, lines 1-15, in particular). The reference humanized MAb 425 (h425) has the following properties: it binds to human EGF-receptor (also known as ErbB1), inhibits the binding of EGF to EGF-receptor, inhibits the EGF-dependent tyrosine kinase activity of the EGF receptor, inhibits the growth of EGF-sensitive cells (see col. 5, lines 23-30, in particular). The reference humanized monoclonal antibody such as h425 is identical to the humanized MAb (h425) in the claimed pharmaceutical composition and is useful for treating cancer in human (see col. 21 through col. 22, col. 5, lines 30, in particular). The '864 patent teaches the reference antibody can be combine with cytokine such as interferon-gamma or tumor necrosis factor (see col. 21, lines 52-56, in particular).

Therefore, it would have been obvious to one of ordinary skill in the art at the time the invention was made to substitute the humanized anti-HER2 mAb 4D5 that binds to (ErbB2) in the pharmaceutical composition as taught by Ye et al for the humanized monoclonal antibody such as h425 that binds to EGF receptor (also known as ErbB1) derived from murine monoclonal antibody MAb 425 for treatment of human tumor as taught by the '864 patent to form a third pharmaceutical composition comprising a humanized monoclonal antibody is MAb 425 (h425) that binds to a different epitope on the ErbB1 receptor and chimeric MAb 225 (IMC-c225) binds to EGF receptor for the same purpose of treating cancer in humans. The term "comprising" is open-ended. It expands the claimed composition to include additional agent.

It also would have been obvious to one of ordinary skill in the art at the time the invention was made to combine the chimeric anti-EGF receptor mAb C225 of Mendelsohn et al with the humanized MAb 425 that binds to different epitope on ErbB1 receptor for treatment of human tumor as taught by the '864 patent to form a third pharmaceutical composition comprising

a humanized monoclonal antibody is MAb 425 (h425) that binds to a different epitope on the ErbB1 receptor and chimeric MAb 225 (IMC-c225) binds to EGF receptor for the same purpose of treating cancer in humans. The term “comprising” is open-ended. It expands the claimed composition to include additional agent.

One having ordinary skill in the art would have been motivated with the expectation of success to substitute because Ye et al teach it is reasonable to combine various monoclonal antibodies in order to inhibit the proliferation of cancer cells stimulated by EGF receptor signals since EGF receptors can be activated by either EGF receptor (erbB1) homodimerization (ErbB1/erbB1) or heterodimerization (erbB1/erbB2) (see page 736, col. 1, in particular).

One having ordinary skill in the art would have been motivated with the expectation of success to combine the humanized monoclonal antibody such as h425 that binds to EGF receptor (also known as ErbB1) as taught by the ‘864 patent with the MAb C225 that binds to EGF receptor (ErbB1) because MAb C225 can act as a tyrosine kinase inhibitor specific for EGF receptor without the side effect of diarrhea with the oral tyrosine kinase inhibitor as taught by Mendelsohn et al (see page 6552, col. 2, in particular).

The ‘864 patent teaches the advantages of using humanized antibody h425 are that the humanized is less likely than either mouse 425 antibodies to raise an immune response in humans and more efficacious when used therapeutically in humans than either the mouse or chimeric 425 antibodies since the humanized antibody has a longer half-life in humans and the least likely to arise adverse immune response in human patient with tumor as a result of repeated use (see col. 22, lines 1-15, in particular).

Given the examination guidelines for determining obviousness under 35 U.S.C. 103 in view of the Supreme Court decision in *KSR International Co. V. Teleflex Inc.* 82 USPQ2d 1385 (2007) and the Examination Guidelines set forth in the Federal Register (Vol. 72, No. 195, October 10, 2007) and incorporated recently into the MPEP (Revision 6, September 2007), the following rationales to support rejection under 35 U.S.C. 103(a) are noted:

- A) Combining prior art elements according known methods to yield predictable results.
- B) Simple substitution of one known element for another to obtain predictable results.
- C) “Obvious to try” --- choosing from a finite number of identified, predictable solutions, with a reasonable expectation of success.

D) Some teachings, suggestion, or motivation in the prior art that would lead to one of ordinary skill to modify the prior art reference to arrive at the claimed invention.

Since combining one known antibody such as chimeric anti-EGF receptor mAb C225 for treating cancer with another known antibody humanized monoclonal antibody such as h425 that binds to EGF receptor (also known as ErbB1) is desirable and have been predictable at the time the invention was made, there would have been reasonable expectation of success in combine the references teachings to arrive at the claimed invention. The motivation to combine can arise from the expectation that the prior art elements will perform their expected functions to achieve their expected results when combine for their common known purpose. Section MPEP 2144.07. It is prima facie obvious to combine two compositions each of which is taught by prior art to be useful for same purpose in order to form third composition that is to be used for very same purpose; idea of combining them flows logically from their having been individually taught in prior art. In re Kerkhoven, 205 USPQ 1069, CCPA 1980. See MPEP 2144.06. The strongest rationale for combining references is a recognition in the art that some advantage or expected beneficial result would have been produced by their combination. This recognition may be an expressed statement in a reference, an implication that can be drawn from one or more references or a convincing line or reasoning based upon established principles or legal precedent.

An obviousness is not the result of a rigid formula disassociated from the consideration of the facts of a case. Indeed, the common sense of those skilled in the art demonstrates why some combinations would have been obvious where others would not. See *KSR International Co. V. Teleflex Inc.* 82 USPQ2d 1385 (2007). From the combined teachings of the references, it is apparent that one of ordinary skill in the art would have had a reasonable expectation of success in producing the claimed invention.

Claim 33 is included in this rejection because the reference antibody obviously binds to the natural ligand binding domain of the receptor because the '864 patent teaches the humanized antibody h245 inhibits binding of EGF to EGF receptor (see col. 5, line 26, in particular). Further, Mendelsohn et al teach the chimeric C225 antibody competes with ligand binding to the ErbB1 (EGF) receptor with high affinity, see page 6552, col. 1, in particular).

Claim 34 is included in this rejection because it is expected that the combination of two antibodies that bind to different epitopes on same ErbB1 receptor is expected to enhance the aggregation of receptors or dimerization of the ErbB1 receptor since Mendelsohn et al teach MAB

225 induces antibody-mediated receptor dimerization without activation of the tyrosine kinase (see page 6552, col. 1, in particular).

It is commonsense that the two antibodies must bind to different epitopes on the same ErbB1 receptor because otherwise the two antibodies would compete with each other for binding if both bind to the same epitope on the same ErbB1 receptor.

Given the structure of antibodies in the claimed composition are the same as that of the prior arts, products of identical chemical composition cannot have mutually exclusive properties. A chemical composition and its properties are inseparable. Therefore, if the prior art teaches the identical chemical structure, the properties applicant discloses and/or claims are necessarily present. In re Spada 15 USPQ2d 1655, 1658 (Fed. Cir. 1990). See MPEP 2112.01.

Applicants' arguments filed January 5, 2009 and December 3, 2007 have been fully considered but are not found persuasive.

Applicants' position is that applicant's disclosure of unexpected properties of the claimed molecules when utilized together, it is submitted that this rejection cannot stand. For example, the Examiner is requested to review the Examples 1-5 provided at pages 41-44 of the instant specification, wherein it is expressly stated that a composition comprising the two antibodies, cetuximab (C225 antibody) and EMD 72 000 (MAb425), results in increased binding to cell-surface receptors (per cell), enhanced cell aggregation, enhanced inhibition of ligand-binding to cognate EGF receptor(s), enhanced displacement of bound ligands, and increased receptor internalization. See, also the disclosure contained in Figs. 1-5 and the description thereof at page 41 of the specification.

In response, the results are expected when combined since the properties of each claimed chimeric antibody C225 (now known as cetuximab) and humanized MAb 425 (h425) (also known as EMD 72000 or MAb425) are known in the art.

For example, at the time of filing, Mendelsohn et al teach chimeric MAb 225 (IMC-c225) binds to EGF receptor with higher affinity ($K_d = 0.39$) than the murine MAb and the natural ligands, (see page 6552, col. 1, in particular) and is useful for treating cancer in humans; MAb C225 is acting as a tyrosine kinase inhibitor specific for EGF receptor (see page 6552, col. 2, in particular) and MAb 225 induces antibody-mediated receptor dimerization without activation of the tyrosine kinase (see page 6552, col. 1, in particular). Ye et al teach the chimeric

antibody C225 competes with EGF for receptor binding and has been shown that repeatedly administered to patient for up to 3 months without eliciting a host anti-antibody response in clinical trial (see page 731, col. 2, second paragraph, in particular).

The '864 patent teaches humanized MAb 425 (h425) has the following properties: it binds to human EGF-receptor (also known as ErbB1), inhibits the binding of EGF to EGF-receptor, inhibits the EGF-dependent tyrosine kinase activity of the EGF receptor, inhibits the growth of EGF-sensitive cells and is useful for treating cancer in humans (see col. 5, lines 23-30, in particular). The combined use of the two antibodies in a pharmaceutical composition is expected to inhibit tumor cell growth more effectively than either antibody alone given that each antibody binds to different epitope on the ErbB1 receptor and each antibody has been shown to inhibit tumor growth.

The increased of binding of antibodies to cell-surface receptors (per cell) and enhanced displacement of bound ligands are expected because is common sense that when two different antibodies bind to two different epitopes located on the same ErbB1 receptor within the erbB1 ligand binding domain, it is expected that more antibodies could be bound per receptor, thus per cell basis as compared to two different antibodies that bind to the same epitope (competing antibodies) on the same receptor. Because the binding of two different antibodies to two different epitope located within the erbB1 receptor ligand binding domain (non-competing with each other) but compete with the natural ligand EGF for binding to EGFR (also known as ErbB1), it is expected that antibody C225 competes with EGF for receptor binding and acting as a tyrosine kinase inhibitor as taught by Ye et al while the humanized MAb 425 (h425) also binds to human EGF-receptor, thereby inhibiting the binding of EGF to EGF-receptor, inhibits the EGF-dependent tyrosine kinase activity of the EGF receptor, inhibits the growth of EGF-sensitive cells as taught by the '864 patent (see col. 5, lines 23-30, in particular).

Since more antibodies can be bound to the same ErbB1 receptor, it is expected that more cell aggregation can result from more antibodies being bound to the ErbB1 receptor (crosslinking) and thereby induces receptor dimerization and receptor internalization.

Because the two antibodies are known to compete with EGF for binding to its receptor, it is expected that less EGF will be available for binding to its cognate EGFR receptors (ErbB1) and thereby enhance inhibition of the EGFR.

Because the two antibodies bind to EGF with high affinity, i.e., Mendelsohn et al teach chimeric MAb 225 (IMC-c225) binds to EGF receptor (also known as ErbB1) with higher affinity

($K_d = 0.39$) compared with EGF binding to its receptor with lower affinity, it is reasonable to expect that the combination of the two antibodies will enhance the displacement of the bound EGF from its receptor.

9. Claim 39 is rejected under 35 U.S.C. 103(a) as being unpatentable over Ye et al (of record, Oncogene 18: 731-738, 1999; PTO 892) or Mendelsohn et al (newly cited, Oncogene 19: 6550,6565, 2000; PTO 892) each in view of US Pat No 5,558,864 (of record, issued Sept 24, 1996; PTO 892) as applied to claims 30, 33-38 and 40 and further in view of US Pat No 6,342,219 (of record, filed April 28, 2000; PTO 892).

The combined teachings of Ye et al and the '864 patent or Mendelsohn et al and the '864 patent have been discussed supra.

The invention in claim 39 differs from the teachings of the combined references only in that a kit comprising the pharmaceutical composition of claim 30 in one package and a carrier in a second package.

The '219 patent teaches a pharmaceutical kit comprising distinct containers for each desired agent where combined therapeutics are provided (see col. 102, lines 5-31, in particular). The '219 patent teaches such kit contains all the necessary reagents and means for commercial sale for treating cancer (see col. 102, lines 52-63, in particular). The kit may contain antibody and other reagent separately (see col. 102, lines 12-25, in particular).

Therefore, it would have been obvious to one of ordinary skill in the art at the time the invention was made to put the pharmaceutical composition comprising chimeric MAb 225 (IMC-c225) binds to an epitope located on the EGF receptor (ErbB1) and humanized MAb 425 (h425) that binds to different epitope on the same EGF receptor (ErbB1) as taught by Ye et al and the '864 patent or Mendelsohn et al and the '864 patent in a first package and a separate package for any reagent as desired as taught by the '219 patent.

One would have been motivated, with a reasonable expectation of success to do this for convenience and commercial expedience. A kit will allow for ease of use for the practitioner since all the necessary reagents, standard and instructions for use are included in a kit as taught by '219 patent (See column 102, lines 41-61, in particular). It is within the purview of one of ordinary skill in the pharmaceutical art to put any carrier in a separate package for stability and convenience of the practitioner as taught by the '219 patent (see col. 102, lines 12-25, in

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particular). From the teaching of the references, it is apparent that one of ordinary skill in the art would have had a reasonable expectation of success in producing the claimed invention.

10. No claim is allowed.
11. Any inquiry concerning this communication or earlier communications from the examiner should be directed to Phuong Huynh, Ph.D. whose telephone number is (571) 272-0846. The examiner can normally be reached Monday through Thursday from 9:00 a.m. to 6:30 p.m. and alternate Friday from 9:00 a.m. to 5:30 p.m. A message may be left on the examiner's voice mail service. If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Eileen B O'Hara can be reached on (571) 272-0878. The IFW official Fax number is (571) 273-8300.
12. Any information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see <http://pair-direct.uspto.gov>. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free).

/Phuong Huynh/

Primary Examiner, Art Unit 1644

February 27, 2009